WR GRACE COMMENTS ON MAY 2, 2002 ACTION MEMORANDUM AMENDMENT AND SUPPLEMENTAL ADMINISTRATIVE RECORD NO. 2, AND SUPPLEMENT TO COMMENTS ON THE ORIGINAL AND SUPPLEMENTAL ADMINISTRATIVE RECORDS

Introduction

WR Grace submits these comments on EPA's Action Memorandum Amendment, dated May 2, 2002 ("May 2, 2002 Amendment"), and Supplemental Administrative Record No. 2. In compiling Region 8's Supplemental Administrative Record No. 2 to support the May 2, 2002 Amendment, EPA continues to avoid the central issue in this case -- whether exposures were occurring in 1999 and afterwards ("current day Libby") that were creating an unacceptable risk to the Libby community that would justify the massive expenditures incurred by the United States. The sampling and analytical data generated by EPA and risks associated with that data fail to support the emergency measures taken by EPA. Region 8 diverts attention from the key issue -- the risk of exposures created by current day Libby -- by discussing studies and other information involving exposure intensities that are dramatically higher than any being experienced by current day Libby. The asbestos-related disease and death in Libby are tragic, and WR Grace's donations to the hospital and creation of a health care plan are attempts to assist sick residents who have been historically exposed to high levels of asbestos.[1] EPA's use of information regarding morbidity and mortality to support its emergency removal actions, however, is inappropriate because conditions causing such disease no longer exist.

Grace's comments are also being submitted to supplement its December 21, 2001 Comments on EPA's Action Memorandum Amendment, dated July 20, 2001, and on the

[1] See Health Care Network presentation (495514); Grace Announces Sweeping Health Care Program for Libby, Montana (495543); St. John's Receives Donation from Grace (495562).

Supplemental Administrative Record. Region 8's June 4, 2002 response to Grace's comments ("EPA June 4, 2002 Document") includes significant information that was not in the Supplemental Administrative Record. Grace, therefore, should have the opportunity to respond. The issues addressed in these comments also relate to the original Action Memorandum, dated May 23, 2000, and original administrative record. Subsequent to the availability of the original administrative record, significant new information is now available that warrants inclusion of these comments into the original administrative record. Grace also incorporates by reference into these comments on the May 2, 2002 Amendment, its December 21, 2001 comments and its comments dated April 29, 2002 on the proposal to add Libby to the National Priorities List.

EPA's Sampling and Analysis

EPA's sampling and analytical data do not support the removal actions conducted by Region 8. As set forth in the attached report by Dr. Richard J. Lee of the RJ Lee Group, Inc. (attachment 1[2]), EPA failed to select the appropriate analytical methods to determine exposures. It also failed to adhere to its own Quality Assurance Project Plans or basic principals of laboratory analysis. Further, EPA biased exposure estimates, and therefore the risk screening assessments, by including overloaded samples, counting inappropriate amphibole structures, misidentifying gypsum, vermiculite, talc, and mica asbestos, and including non-respirable particles. Comparison of the values reported as sample concentrations by Dr. Weis with the results of the samples in the database indicates that Dr. Weis used incorrect concentrations in calculating risk. Region 8 also failed to reach the necessary analysis level to support its trigger level on time-critical intervention. Region 8 miscalculated the asbestos concentrations and failed to use the NIOSH 7402 method to relate the TEM estimates of exposure to PCM values.

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[2] Attachment 1 includes Dr. Richard J. Lee's report as well as 92 supporting documents.

Region 8 further inappropriately included cleavage fragments in its exposure estimates and risk assessments. It cherry-picked certain samples and excluded other samples, resulting in an overestimation of asbestos exposure and therefore risk to residents of current day Libby.

As explained in detail in Dr. Lee's report, the use of indirect preparation results in an overestimation of counts. The use of this type of preparation by Region 8 contractors resulted in an overestimation of asbestos counts by at least an order of magnitude.

Approximately 74 percent of EPA's analytical results include the improper counting of cleavage fragments. Cleavage fragments do not contribute to risk and are forbidden to be counted by applicable regulations. OSHA's rulemaking in 1992 evaluated whether cleavage fragments should be counted as asbestos and concluded that the evidence does not support regulating such fragments as asbestos. 57 Fed. Reg. 24310 (June 8, 1992). The applicable methods for analyzing samples also do not allow cleavage fragments to be counted. In addition, the IRIS methodology dictates that only asbestos be counted. Moreover, EPA cannot simply assume, as Dr. Weis does in his April 24, 2002 memorandum, that "if cleavage fragments do constitute a significant fraction of Libby structures, then it is likely they also constitute a significant fraction of the structures the miners breathed." As set forth below, the fiber dimensions the miners breathed were longer and thinner and in far greater quantities than the fibers being found in Libby today.

Dr. Lee's report and the report of Dr. Elizabeth L. Anderson of Sciences International, Inc. (attachment 2[3]) conclude that the scientific evidence does not support any conclusion that cleavage fragments are carcinogenic, based on review of human and animal studies. They further point to the conclusion of U.S. regulatory agencies, their scientific counterparts, and the

[3] Attachment 2 includes Dr. Elizabeth L. Anderson's report, as well as 26 supporting documents.

International Agency for Research on Cancer which have all uniformly concluded that cleavage fragments and non-asbestiform substances are not considered carcinogenic agents and are not regulated as posing a health risk.

Region 8 is also including non-asbestiform minerals, winchite and richerite, in its counts. Dr. Lee estimates that at least 15 percent of Libby amphiboles are not regulated amphiboles. According to Kathleen Rest, Acting Director, NIOSH, in comments before the United States Committee on Health, Education, Labor and Pensions (July 31, 2001) (495841), 80 to 90 percent of fiber contaminant in vermiculite is not currently regulated as asbestos. In his July 31, 2001, statement before the Committee on Health, Education, Labor, and Pensions, United States Senate (495786), R. Davis Layne, Acting Assistant Secretary of Labor for OSHA, stated that "[i]n 1992, OSHA reviewed available relevant evidence concerning the health effects of nonasbestiform tremolite . . . OSHA determined that there was insufficient evidence to support a finding that exposed workers would be at a significant risk from these substances if they were not regulated in the asbestos standard." The cleavage fragments and non-asbestiform minerals are not hazardous substances as defined by section 101 of CERCLA.[4]

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[4] See 40 C.F.R. § 61.141 (asbestos means the asbestiform varieties of serpentinite (chrysotile), riebeckite (crocidolite), cummingtonite-grunerite, anthophyllite, and actinolite–tremolite). See also July 30, 2001 Workplace Exposure to Asbestos, Testimony before the Senate Committee on Health, Education, Labor and Pensions (495603) (definition for asbestos does not include cleavage fragments or solid solution series for six asbestos minerals); Meeker, The Chemical Composition and Physical Properties of Amphibole from Libby, Montana, A Progress Report (487400) (winchite, richerite, ferro-edenite and magnesio-arfvedsonite are not regulated asbestos); EPA Region 10, Asbestos and Your Health (495592) (asbestos is a name applied to six minerals (amosite, chrysotile, tremolite, actinolite, anthophyllite, and crocidolite)); Brandli, Characterization of Amphibole and Amphibole-Asbestos from the Former Vermiculte Mine in Libby Montana (May 2002) (495958) (Recent work by Wylie and Verkouteren (2000) and Gunter et al. (2002) shows that the amphibole minerals present are actually winchite and richterite; Libby vermiculite mine has no tremolite); August 23, 2001 Year 2000 Medical Testing of Individuals Potentially Exposed to Asbestiform Minerals Associated with Vermiculite in Libby to the Community (ore primarily contaminated with winchite and richerite). Much of EPA's removal actions have not addressed asbestos at all, but rather mineral particles that

Aside from the cleavage fragments and the non-asbestiform minerals counted by EPA, 12 percent of the counts include amphiboles that were not in the data, based on improper EDS spectra.

When Dr. Weis' calculations are redone with the appropriate concentrations, the exposures are at or below background concentrations for ordinary living and cleaning activities, and below the current permissible exposure values for other activities. These exposures are further reduced when the presence of cleavage particles and misidentified particles are taken into account.

Exposure Pathways

While EPA has conducted an enormous sampling project in Libby, the results used by EPA to support its removal actions have marginal relevance to the removal actions conducted. EPA states that:

> Sample results for sweeping, transferring vermiculite between containers, and EPA cleanup activities yielded airborne asbestos levels of over 1.0 f/cc. Sampling for recreational activities have shown exposure levels of 0.2 f/cc on the track.

May 2, 2002 Amendment at p. 9. Two of these scenarios, transferring vermiculite between containers and sampling for recreational activities, are historical and irrelevant to current day Libby. With respect to "transferring vermiculite between containers," Region 8 is apparently referring to a test conducted in 1982 regarding the transfer of 400 pounds of vermiculite to a container under artificial, worst case test conditions. Grace's December 21, 2001 letter to Mr. Cohn explained why that test does not relate to conditions in current day Libby. With

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Region 8 refers to as Libby Amphiboles. See CDM, Final Sampling and Analysis Plan, Remedial Investigation Contaminant Screening Study (April 2002) (495637) ("These removal actions [screening and export plants, the Flyway, KDC Bluffs, Plummer Elementary, Libby High School, Libby Middle School, and several residential and commercial properties] are designed to remove major sources of Libby amphibole (LA) in and around the city of Libby").

respect to the track, Region 8 apparently refers to a Grace memorandum, dated July 27, 1981, indicating that the high school tracks showed 0.14 f/cc and 0.22 f/cc for two test runners on the track. For that reason, Grace "planned to remove and replace the material expeditiously," as set out in that memorandum. Grace offered to either remove the vermiculite or pave over it, and the school district chose paving.[5] In contrast to these historical exposures, no children are running on tracks at these historical levels in current day Libby. EPA cannot support any allegation that these historical levels approximate levels found in EPA's sampling.

Aside from the specific scenario of the track, current day Libby differs dramatically from conditions in Libby before 1990 when the mine closed. The vermiculite mining operations had a dry mill until 1974, and conditions at the dry mill were very dusty.[6] A wet mill was installed in 1974[7] and was fully operational by 1976.[8] At a screening plant in another location, the ore was graded, transported by conveyor belt across the Kootenai River, and shipped by rail to other processing plants.[9] An exfoliation plant also operated in Libby from 1920 to 1949, when it was demolished.[10] Another expansion operation occurred at the former export plant, starting in the

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5 See Montanian, EPA Seals School Skating Rink (April 18, 2001) (495386); May 9, 2001 Article (495874). Had EPA provided Grace the opportunity, Grace would have cleaned up surface vermiculite at the schools, whether or not it was creating an unacceptable risk. Unfortunately, Region 8 has failed to give Grace the opportunity to perform any of the cleanup, other than the Export Plant.

6 April 19, 1962 Report on Industrial Hygiene Study of Zonolite Company by Benjamin Wake (335261).

7 The United States has the mistaken impression that the dry mill operated with the wet mill until 1985. See CDM, Final Sampling and Analysis Plan, Remedial Investigation Contaminant Screening Study (April 2002) (495637). The dry mill was no longer used at all after the wet mill came fully on line.

8 Memorandum from H. A. Eschenbach to F. E. Bona, dated July 28, 1978 (492100).

9 March 31, 2001 Inspector General Report (495722).

10 Id.

1950's.[11] Expansion at this plant ceased sometime around 1970, although the area was still occasionally used to bag and export milled ore until mining operations stopped in 1990. ATSDR also noted that alternative pathways for historical exposures might be related to elevated concentrations in ambient air and recreational exposures to children playing in piles of vermiculite.[12] If EPA's allegations are correct regarding the high historical ambient exposures before the mine closed in 1990, these allegations emphasize the difference in ambient air quality between historical and current day Libby. EPA refers to Mr. Eschenbach's discussion of historical fibers, a "Source Emissions" document dated 1975, and the MRI (1982) document to support EPA's views of historically high ambient exposures.[13] Another document alleges that EPA tested air outside of Libby in 1980, and these air samples from outside town showed levels of asbestos as high as 0.5 f/cc.[14] According to this document, "[w]hile information about past exposure is limited, we believe that these were not isolated instances and that during dry weather times and during weather inversions over Libby or during periods of high production at the mine or processing area, asbestos fiber counts could have been much higher." In contrast, Region 8 has emphasized that ambient levels in current day Libby do not show ambient levels of concern.[15]

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11 Id.

12 February 22, 2001 Preliminary Findings of Medical Testing of Individuals Potentially Exposed to Asbestiform Minerals Associated With Vermiculite in Libby, Montana: An Interim Report for Community Health Planning (February 22, 2001) (487402).

13 June 4, 2002 EPA Document at p. 27.

14 Public Notification of Past Asbestos Exposure in Libby (#2) (485300).

15 March 14, 2002 CAG Meeting Summary (495610) ("air monitoring results are negative for asbestos contamination"); Ask EPA, October 8, 2001 (495700) (". . . EPA has found no evidence of asbestos exposures (or risks) from ambient (outdoor) air around town . . .")

Logic defies EPA's allegation that a massive soil removal program would generate the same levels as "activities normally undertaken by residents such as digging, rototilling, sweeping or driving over contaminated media."[16] Dr. Weis' December 20, 2001 memorandum simply does not have the data to support releases of concern from driveways, garden soils, and yards. Apparently, EPA staff raised similar concerns with Dr. Weis' discussions.[17]

If the May 2, 2002 Amendment is referring to the workers who simulated sweeping in the Export Plant and Screening Plant, Dr. Lee's report indicates that Region 8's sampling results are flawed because most of the samples used an indirect preparation method that violates standard EPA procedures and one sample is suspect.

Many of the documents in Supplemental Administrative Record No. 2 indicate that asbestos levels in soils should not be used to predict airborne levels, as Grace has been asserting from the beginning of its communications with Region 8. For example, the document, Health-Based Soil Investigation Levels (495604), states:

> No relationship between soil levels and air levels can be predicted for an asbestos-contaminated site. The Addison et al. study shows trends using a laboratory test system but the high amount of variability which may exist on contaminated sites makes it difficult and probably inappropriate to apply results of this study to contaminated sites.

Grace also disagrees strongly with the implication of EPA's contention that "insulation, just like other Libby vermiculite media, creates high airborne levels of amphibole asbestos when disturbed by human activities."[18] Grace studies on vermiculite insulation and EPA's Office of

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16 EPA June 4, 2002 Document at p. 29.

17 March 20, 2002 Memorandum from Bill Brattin to Chris Weis (495594).

18 EPA June 4, 2002 Document at p. 30. As a result of recent discussions with the United States, these comments do not focus on insulation. At the appropriate time and forum, Grace will address such documents as Final Report, Site Assessment, Vermiculite Removal Building E-12, C.F.B. Shilo, Shilo, Manitoba (April 3, 1997), involving a large demolition product of a Canadian building.

Pollution Prevention and Toxic Substances (OPPT) indicate that normal activities at homes with insulation do not result in unacceptable risks.[19] According to the OPPT Memorandum commenting on the May 2, 2002 Amendment draft (495605), "the data we have at this point do not appear to suggest that ZAI will contribute significantly to risk in a typical attic situation . . .".

Historical v. Current Fibers

The fibers found in EPA's sampling have a different fiber dimension than the fibers that caused disease in miners. In his April 24, 2002 memorandum, Dr. Weis similarly acknowledged this issue, stating that "[o]ne important issue we are evaluating is whether or not the fibers the residents encounter in the residential and commercial areas of Libby today are similar to those that are known to have caused disease in miners." As set forth in Dr. Lee's report, fibers found in current day Libby are shorter and fatter than the longer and thinner fibers found to cause illness in the Libby miners. EPA's contractors themselves have indicated that "most of the fibers detected on this project have been under 5 μm in length."[20] This information refutes one of Region 8's "theoretical explanations" that "shorter fibers in fact are contributing to toxicity that has been readily observed in Libby."[21]

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[19] See Comments by WR Grace on Proposal to Add Libby, Montana to the National Priorities List, dated April 29, 2002 and exhibits, which are incorporated by reference, including Health Risks from Exposure to Zonolite Home Insulation; Critical Evaluation of the Scientific Evidence Available from the ATSDR Studies in Libby, Montana, by Gary M. Marsh, Ph.D., Professor of Biostatistics, Graduate School of Public Health (495531); February 28, 2002 CAG Meeting Summary (495608) ("The unit of EPA that regulates asbestos, the Office of Pollution Prevention and Toxic Substances, recently came out in opposition to the insulation removal in Libby.").

[20] Memorandum from Mark Raney to Jeanne Orr, dated May 7, 2002 (495623).

[21] EPA June 4, 2002 Document at p. 33.

The accepted scientific view is that long, thin, durable fibers are more potent in causing disease.[22] Region 8's own administrative record is replete with studies supporting that view, Dr. Weis has advanced that position, and papers at recent conferences support that position.[23] EPA admits in its June 4, 2002 Document that there is a "relatively small fraction of long fibers observed by EPA and Grace."[24]

Region 8 tries to avoid the logical conclusion that the short fibers found in the sampling indicate lower toxicity by an alternative "theoretical explanation" that "the relatively small fraction of long fibers observed by EPA and Grace are in fact extremely potent."[25] Dr. Suresh H. Moolgavkar, a noted biostatistician and epidemiologist, has addressed the issue of potency by

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22 See Dr. Anderson Report.

23 See , e.g., Cotran, Pathologic Basis of Disease (1999) (495533) ("The length of amphibole fiber also plays a role in pathogenicity, those longer than 8 mm and thinner than 0.5 mm being more injurious than shorter, thicker ones"); Weis, Residual Mineral Fiber Contamination at the Former W.R. Grace Screening Plant and Export Plant Poses an Imminent and Substantial Endangerment to Public Health (May 17, 2000), (337945) ("Fiber size length and width may influence toxicity. Clearance of fibers from the lung is inhibited and fiber toxicity is significantly enhanced when fiber length is greater than approximately 8 μm (Blake et al., 1998)"); Weis, Amphibole Mineral Fibers in Source Materials in Residential and Commercial Areas of Libby Pose an Imminent and Substantial Endangerment to Public Health (Dec. 20, 2001) (495671) ("Although this risk model has not yet been peer reviewed, it is potentially important because fiber toxicity is expected to vary as a function of fiber length, with longer fibers displaying greater toxicity than shorter fibers"); Churg, Lung Asbestos Content in Long Term Resident of a Chrysotile Mine Town (1985) (338278) (". . . there is general agreement that long fibers of asbestos have, on a one for one basis, more potential to cause disease than short fibers."); Presentation by Kenneth Donaldson, Biomedicine Research Group, Napier University, Edinburgh, entitled "The Role of Fiber Length in Inflammation and Disease, at Asbestos Conference June 24, 2002 in Missoula, Montana, entitled "2002 New Directions and Needs in Asbestos Research," Center for Environmental Health; Presentation by Vincent Castranova, National Institute for Occupational Safety and Health, Role of Fiber Length in Cytotoxicity and in the Activation of Macrophages, presented at same conference.

24 EPA June 4, 2002 Document at p. 33.

25 Id.

analyzing the Amandus and McDonald cohorts and has found that the potency is lower than the IRIS potency used by Dr. Weis in his screening level calculations.[26] See attachment 3.

Dr. Weis' speculations about whether his screening risk calculations underestimate risk should be completely disregarded. Even EPA staff raised questions about Dr. Weis' statements regarding his concern about the short fiber sizes found by the sampling.[27]

Therefore, the fiber dimension data indicate that the risk estimates by Dr. Weis are overestimated, not underestimated as alleged by Dr. Weis, because (1) the environmental fibers are shorter and wider than the fibers causing the disease described by Amandus, and (2) the potency of Libby fibers are less than the IRIS potency estimate used by Dr. Weis in his screening level calculations.

Risk Analysis

As described in detail in Dr. Anderson's report, Dr. Weis' screening risk evaluations are obscure, contain numerous apparent errors, and cannot be duplicated. The lack of transparency of his work conflicts with EPA's guidelines calling for EPA risk evaluations to be under-

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[26] EPA's allegation in its June 4, 2002 Document that "NIOSH . . . found that tremolite exposures associated with mining in Libby may be among the most potent exposures related to asbestos risks for mesothelioma" is an exaggerated interpretation of Stayner et al. "Occupational Exposure to Chrysotile Asbestos and Cancer Risk: A Review of the Amphibole Hypothesis (487096). Stayner was comparing the 1986 McDonald study of Libby miners to a study of Quebec miners and millers. He was not purporting to conduct an exhaustive review of the potency of different amphiboles in causing mesothelioma. Even Dr. Whitehouse, who treats patients with asbestos-related disease from Libby, is more cautious than Region 8 in stating that "McDonald (1999) speculated on tremolite's increased fibrogenicity, and it would appear that tremolite is much more fibrogenic than chrysotile, and possibly more so than other amphiboles as well." Draft Report entitled Asbestos Related Pleural Disease Due to Tremolite Causes Progressive Loss of Lung Function (495889).

[27] Memo from Bill Brattin to Chris Weis dated March 20, 2002 (495594) ("... some researchers have argued that toxicity is highest for long thin fibers. This further weakens the points about potential toxicity of non-PCME fibers made in the preceding paragraph.").

standable. Dr. Weis' assessments also include unreasonable assumptions and statements about potential risks in Libby, and overstate the current risk to residents.

Dr. Anderson has performed her own risk assessment using the more accurate analytical results provided by Dr. Lee. These indicate that risks existing today in Libby are much lower than estimated by EPA.

In addition, Dr. William J. Hughson's report (attachment 4[28]) states that there is substantial evidence in the medical literature indicating that EPA's linear no-threshold model is not correct and that substantial amounts of asbestos are tolerated in human beings with no evidence of adverse health effects.

Mortality Study

As Grace has previously commented, the ATSDR mortality study is irrelevant to the issue of whether exposures were occurring in 1999 when Region 8 started its investigations in Libby because the mortality was caused by historically much higher exposure levels. According to the report, the period selected for the death certificate review was to account for the period of highest exposures, "based on reports indicating that peak production volumes occurred during this period and engineering controls were being implemented during or after this period." EPA admits that the mortality relates only to historic exposures, but makes the unsubstantiated leap that "similar levels of exposure [as occupational exposures] may be occurring routinely in this community," failing to cite a single reference.[29] The sampling data from 1999 to the present simply do not support EPA's speculation.

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[28] Attachment 4 includes Dr. William J. Hughson's report, as well as seven supporting documents.

[29] June 4, 2002 Document at p. 9.

EPA alleges in its June 4, 2002 Document that an update of the mortality study shows that asbestosis rates are substantially higher and lung cancer is statistically elevated over expected rates, but Region 8's Supplemental Administrative Record No. 2 fails to include this information. Grace has also requested this information in discovery, but the United States to date has not provided it. Therefore, Grace cannot comment on it at this time. Assuming the information alleged by EPA is correct, the question again is whether exposures were occurring in 1999 in Libby that were similar to those that occurred during the historical time period that caused this mortality. There is no evidence that such is the case.

Health Screening Study

Grace has not been provided with the combined medical screening results from the summer of 2000 and the summer of 2001 to arrive at a new data set, referenced in EPA's June 4, 2002 Document, and therefore lacks the ability to comment on it. Moreover, Grace has not been provided with the medical information, such as chest x-rays and individual B reader data, of either the 2000 or 2001 results that would allow it to independently evaluate the screening results. Grace therefore can comment now only on the information available to it.

If EPA is referring to the screening study in asserting that "chronic exposure to high levels of asbestos has compromised the health of many Libby residents," EPA has conceded that "disease present today in Libby was likely caused by exposures during the 60s, 70s and 80s (due to the latency period)."[30] Dr. Hughson's report also indicates that the findings of the screening study relate to exposures occurring decades ago. EPA, for example, refers to the 1 percent of

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[30] EPA June 4, 2002 Document at p. 33. See also ATSDR Launches Medical Testing Program for Residents of the Libby (MT) Area (335464) ("The purpose of the medical testing program is to evaluate the current health status of those people who were present in Libby during the period of highest exposure to asbestos.")

participants in the medical screening program that had parenchymal fibrosis reported by at least two B-readers, but it fails to add that these participants were exposed to historical exposures.[31]

The low percentage of persons identified in the screening study with parenchymal fibrosis is noteworthy. This percentage is below the 3.4% found in the control group in Anderson, Household Exposure to Asbestos and Risk of Subsequent Disease (495425) (Table 7). Dr. Hughson's report states that "[s]ince a large proportion of the study population had either occupational or domestic exposure to asbestos, this low prevalence of interstitial findings is quite reassuring, and certainly does not suggest a risk of interstitial fibrosis in people with low-level ambient or activity-related exposures."

Grace has already commented in its December 21, 2001 comments that the screening study has many limitations, including use of a volunteer study, lack of a control group, reader variability and bias, and the significance of obesity and oblique films.[32] In EPA's June 4, 2002 Document, Region 8 cites four studies comparing Libby to "differing groups within the United

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31 EPA June 4, 2002 Document at p. 23.

32 Lawson, Reliability and Validity of Chest Radiograph Surveillance Programs (495644), supports many of Grace's comments. First, Lawson agrees that the accurate diagnosis of pleural change on x-ray is exceedingly difficult, and that what may appear to be pleural changes are in reality subpleural fat or muscle that is entirely normal. This is precisely why the high level of obesity (BMI) in the Libby population is so relevant. It dramatically increases the risk that what the screening study readers were actually seeing was subpleural fat, not pleural thickening. Second, the study authors point to the relevance of control x-rays -- their study design required that 25% of the x-rays read by their B-Readers were from people not exposed to asbestos -- a point also made by Grace. In the ATSDR screening study, all of the readers knew that they were only reading x-rays from Libby residents. The potential for bias in such a situation is well recognized and noted by Lawson and colleagues. This problem is not solved, as EPA suggests, by simply blacking out the individual's names on their x-rays. Finally, Lawson recognizes that regardless of the type of x-ray views used, false positives, as well as negatives, can occur. This is precisely the point made by Grade, that a "positive" x-ray finding during a screening does not mean that an individual has an asbestos related disease.

States believed to have no substantial work-related asbestos exposures."[33] This comparison, however, is unrealistic since the participants in the Libby screening study included those with historical work-related asbestos exposures. Moreover, the first study, Prevalence of Radiographic Appearance of Pneumoconiosis in an Unexposed Blue Collar Population, Castellan (495437), was a study involving asbestosis, not "pleural abnormalities." Notably, two of the three studies that were adequately described involved the standard, and not oblique, chest x-rays.[34] One study also discussed the bias resulting when B-readers were not blind to study design and objective.[35] As Grace previously commented, such controls and precautions against bias were not used in Libby.

The Supplemental Administrative Record No. 2 contains disturbing information that Region 8 has tried to change the language of the ATSDR report, not for scientific reasons, but solely for litigation purposes. An example is Dr. Weis' revisions to the ATSDR draft, where he insisted that ATSDR add as a "goal" of the study "to provide EPA with information needed to

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[33] Grace cannot identify the reference to 1.8 percent as a background rate for pleural abnormalities in EPA's June 4, 2002 Document. EPA June 4, 2002 Document at p. 13. Apparently, this figure was derived from a chart prepared by Aubrey Miller and Robert Castellan showing that 4.6% of controls had pleuropulmonary abnormalities characteristic of asbestos. Grace notes that Hillerdal, Pleural Plaques in the General Population (495541), stated that 3 to 4 percent of the male population in a slightly industrialized county such as Uppsala will have pleural plaques, and Neri, Lesioni Correlate ad Asbesto Rilevate Mediante TC ad alta Definizione di Immagine in Lavoratori Asintomatici (495503), stated that in a control group (20 subjects without any known professional exposure to asbestos), HRCT identified 5 cases with small pleural plaques while only one case presented parenchymal bands.

[34] Castellan; Stibolt, Pulmonary Health Risks Among Northeast Loggers (495487) (involving a high incidence of pleural thickening among loggers, likely related to chest trauma).

[35] Castellan (high prevalence of small capacities of profusion (11%) in chest radiographs from 200 hospitalized patients may be caused by failure to blind B-readers). See also, Anderson, Household Exposure to Asbestos (495425) (chest x-rays of household contacts, factory workers, and controls were intermixed and read without knowledge of exposure category); Lawson, The Reliability and Validity of Chest Radiograph Surveillance Programs, (495644) (involved "control-radiographs from individuals without a known fiber exposure").

identify and eliminate current exposures to asbestos in the community [THIS SECTION IS IMPORTANT FOR YOUR COST RECOVERY — PLEASE DON'T REMOVE]." (495667)

Non-Occupational Case Series

EPA's reliance on the "eight cases of non-occupational asbestos-related lung abnormalities among a subgroup of twenty-two Libby patients being followed by a Spokane pulmonologist (Dr. Whitehouse)" fails to support EPA's argument that the exposures that occurred historically were occurring in 1999.[36] EPA has included only a poster in the Supplemental Administrative Record No. 2 regarding the eight cases. Grace has not been provided with the medical records (history, physician exams, test results) or the review by the expert panel of Dr. Whitehouse's cases and therefore Region 8 precludes Grace's ability to comment meaningfully. As of May 19, 2002, when the poster was presented at the American Thoracic Society, the poster was still undergoing review. Grace's comments are therefore based on the extremely limited amount of information available about the case series.

The case series does not support the conclusion that exposures creating unacceptable risks were occurring in current day Libby. The conclusion on the poster is that "[w]hile living in the Libby area during the period the mine operated is adequate documentation of an individual's potential for exposure to asbestos, confirmed cases of clinical asbestosis typically had one or more 'special' exposure pathways." (Emphasis added.) Therefore, the case series seems to suggest that living in Libby during the time the mine operated was not enough exposure to cause asbestos-related conditions; rather, one or more "special pathways were involved." The case series also points out the confusion created by EPA's allegations that the "69% of those having

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36 EPA June 4, 2002 Document at p. 10.

lung abnormalities [in the screening study] had no occupational exposure at all."[37] ATSDR eliminated from consideration in the case series, not only the persons who actually worked at the mine, but others who were exposed to miners' clothes or who otherwise worked with Zonolite. Of 27 patients of Dr. Whitehouse who were never employed by the mine, 6 patients were removed from consideration as having had household (i.e., exposure through miners' clothes) or secondary contacts, 3 participants were removed as having had occupational exposure to commercial asbestos, and 5 participants were removed as having had occupational exposure to vermiculite from a non-mining job, such as working at the Zonolite dump and selling or delivering Zonolite.[38] Of the 8 participants who the poster authors conclude were "participants without occupational exposure," each participant except for participant 21 played at the ballfield near the exfoliation plant, a pathway which did not exist in 1999. Five of the participants played in and around piles of vermiculite and four popped vermiculite on the stove.[39] According to Dr. Lybarger's presentation at the May 19, 2002 American Thoracic Society meeting regarding the screening study, these were historical pathways. The poster itself states that the:

> contribution of various environmental pathways cannot be fully assessed with the information available, but childhood exposures may be important. For example, 7 of the 8 'environmental' participants reported playing at the ballfields adjacent to the expansion plant in Libby.

The study also did not quantify exposure.

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37 EPA June 4, 2002 Document at p. 10.

38 Dr. Whitehouse similarly referred to patients with exposures that no longer exist in his presentation on February 23, 2000 at the "Libby Asbestos Exposure Scientific Council Meeting" in Cincinnati, Ohio. He referred to "a resident who lived near the processing facility; a logger who worked in the forest near the milling operation; an upholstor (sic) who repaired trucks used at the mining operation; and an individual who delivered diesel fuel to the mill on a regular basis."

39 The poster also states that the total pathway reported did not include the exposure pathways for 'Dust, other job' or 'Asbestos, other job' which is difficult to interpret.

Of the eight patients identified with disease by Dr. Whitehouse, one (case 1) was not confirmed for epidemiologic purposes when the expert panel reviewed the medical information. Significantly, this individual states that he or she was exposed through every "environmental exposure pathway, including 'vermiculite insulation in the home,' 'handled vermiculite insulation,' 'used vermiculite gardening,' 'recreation on Rainy Creek Road,' 'played at ballfield near exfoliation facility,' 'played in and around piles of vermiculite,' and 'popped vermiculite on stove.'" Notwithstanding that this case reportedly involved the most number of exposure pathways, the patient therefore was not confirmed by ATSDR for epidemiological purposes as having asbestosis.

Participant 11 was described in more detail. According to the poster, this participant is in his 70s and was a smoker. During his early years, his family lived within one block of the vermiculite expansion facility. He remembers playing in the storage bins as a child. He also has a family member diagnosed with asbestosis. As of 1999, an expansion plant no longer operated and storage bins no longer existed in Libby. He also was exposed through six pathways, including playing at the ballfield near the exfoliation plant and popping vermiculite on the stove.

The "non-occupational" case series, therefore, does not provide evidence that living in current day Libby would result in asbestos-related abnormalities.

CT Study

Region 8 also refers to the CT study currently being undertaken to evaluate whether persons with normal or questionable radiographic findings have asbestos-related abnormalities.[40] The Supplemental Administrative Record No. 2 does not have this information, and therefore

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[40] EPA June 4, 2002 Document at p. 15.

Grace cannot comment on EPA's allegation that the 18 percent identified in the screening study may be a conservative estimate.

Lockey and Other Studies

Dr. Anderson's report addresses Region 8's allegation that the "medium" exposure group in the Lockey study had an elevated level of "lung abnormalities," and that this group was similar to exposure in the screening and export plants. EPA argues that, therefore, one would expect "lung abnormalities" for people at the screening and export plant. EPA's argument fails for three reasons, as explained in Dr. Anderson's report. First, the medium exposure group was not associated with a statistically elevated level of lung abnormalities. Second, the exposures at the screening and export plants are lower than stated by EPA. Third, the actual exposures at the screening and export plants were significantly lower than the exposures associated by Lockey with lung abnormalities, and were even lower than the control group in the Lockey study.

EPA continues to point to studies with exposures much higher than the levels found by the sampling in current day Libby. For example, EPA's June 4, 2002 Document at p. 16 refers to the study, Long Term Radiological Effects of Short Term Exposure to Amosite Asbestos Among Factory Workers, Ehrlich (487155). That study, which involved posteroanterior chest radiographs, involved "exposures of the magnitude" not likely to "occur often today in industrialised countries." The study, in fact, said that it was not pertinent to the question of concentration threshold since the concentration was "high to extremely high by today's standards."

The report, Wright, et al., Fatal Asbestosis 50 Years After Brief High Intensity Exposure in a Vermiculite Expansion Plant (492243), also involved an intense occupational exposure. The decedent described in this report, Mr. Parent, unloaded vermiculite ore, operated a forklift, shoveled ore into canvas bags, and, occasionally shoveled ore into ovens. He recalled a heavy

dust burden on his clothes and snorting out black dust from his nostrils in the evening after bathing. He subsequently served in the Navy for six years and he smoked cigarettes for approximately 23 years, averaging a pack of cigarettes per day.

In the complaint (attachment 5) filed concerning this case, the plaintiffs alleged that, when in the Navy from 1953 to 1958, Mr. Parent personally handled and worked near large quantities of asbestos-containing materials manufactured by various defendants, including AC&S, Combustion Engineering, Thorpe Insulation, Flintkote, Flintkote Mines Ltd., Bell Asbestos Mines, Atlas Turner, and Union Carbide. In responses to interrogatories (attachment 5), plaintiffs detail the decedent's exposure to asbestos in the Navy. Mr. Parent worked in the boiler rooms and engine rooms of Navy ships and also "removed asbestos to repair and maintain steam and condensated lines, valves, pipes and pumps." Mr. Parent also removed other asbestos-containing products, including insulation, packing materials, pipe coverings, and cement.

Region 8 cannot assume that tremolite from exposure at the California plant is the cause of Mr. Parent's asbestosis when the plaintiffs have alleged long-term, heavy asbestos exposure from the U.S. Navy and the case has not yet been litigated. As set forth in attachment 6, Diffusing the Asbestos Litigation Crisis: The Responsibility of the U.S. Government, by the Washington Legal Foundation (1986), claimants in over one-half of the 35,000 asbestos-related personal injury suits in existence in the mid 1980s alleged that they were exposed to excessive amounts of asbestos in the course of their employment at Government-owned shipyards or at private shipyards engaged in the manufacture and repair of United States naval and merchant vessels pursuant to Government controls.[41] In fact, many of the health studies in the

[41] The Parent litigation follows this pattern. As described in this article, plaintiffs in the asbestos disease suits resulting from past shipyard exposures are caught in a tangle of workmen's compensation and medical regulations that at best afford them arbitrary, inefficient and tardy relief. Accordingly to the article, the Government rests comfortably in the knowledge that

administrative record and produced by the United States appear to address asbestos-related disease from working for the United States.[42]

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federal workers' compensation statutes preclude workers' suits against the Government. As a result, workers have brought suit against the only "available" defendants -- the manufacturers. The manufacturers in turn have brought claims against the United States for indemnification or contribution for the sums the manufacturers have paid to asbestos claimants.

The United States Court of Appeals for the Fifth Circuit, noting the inability of the nations court system to deal effectively or fairly with the matter of compensating individuals injured as a result of asbestos exposure, recently concluded that "a desperate need exists for federal legislation in the field of asbestos litigation," and, according to the article, the United States Government bears a very large share of responsibility for the asbestos tragedy which clearly is the legacy of past extensive shipyard exposures. The article highlights the information available to the United States about asbestos, including amosite, which was never conveyed to workers at Government-owned shipyards and at private shipyards engaged in the manufacture and repair of United States naval and merchant vessels pursuant to government controls. As late as 1983, according to the article, the Navy continued to violate OSHA regulations and its own requirements for handling asbestos safely.

Another article, "Abandoning Ship: Governmental Liability for Shipyard Asbestos Exposures," 67 N.Y.U.L. Rev. 1034 (Nov. 1992) (attachment 7), similarly points out that the government's "failure to protect or even warn shipyard workers is not simply a question of wartime emergency, because recent evidence indicates that the Navy still fails to enforce its own safety standards and still fails to warn workers about the potential dangers of shipyard work." It also points out that federal worker's compensation schemes have denied many workers any compensation. See also Dube v. Pittsburgh Corning, 870 F.2d 790 (1st Cir. 1989) (case involving daughter who died of mesothelioma from exposure to father's clothes during his civilian work for the Navy).

[42] See, e.g., Dust Exposure and Mortality in an American Factory Using Chrysotile, Amosite, and Crocidolite in Mainly Textile Manufacture (1983) (487081); Kane, Malignant Mesothelioma in Young Adults (1989) (495482) (mother and child diagnosed with mesothelioma and father with adenocarcinoma from father's work as shipyard pipe insulator); Spirtas, Malignant Mesothelioma: Progression Attributable to Risk of Asbestos Exposure (1994) (495501); Ehrlich, Long term Radiological Effects of Short Term Exposure to Amosite Among Factory Workers (1992) (487155); Anton-Culver, Immune Response in Shipyard Workers With X-Ray Abnormalities Consistent with Asbestos Exposure (1988); Kilburn, Asbestos Disease in Family Contacts of Shipyard Workers (1985) (344136); Selikoff, Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976 (1979) (486438); Seidman, Mortality Experience of Amosite Asbestos Factory Workers: Dose-Response Relationships 5 to 40 Years After Onset of Short-Term Work Exposures (1986) (485939); Anderson, Asbestosis Among Household Contacts of Asbestos Factory Workers (1978).

EPA asserts that "the majority of medical research of asbestos-related disease and the observed pathology and progression of asbestos-related lung disease in Libby indicate that once exposed, an individual with lung abnormalities has a high probability of manifesting disease and impairment."[43] The screening study, however, found little evidence of progression. The study

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[43] In its June 4, 2002 Document at p. 3, EPA purported to respond to "Grace's characterization of the observed abnormalities as 'beauty spots'," in Grace's December 21, 2001 comments. This quote exemplifies the extent to which Region 8 mischaracterizes Grace's comments, presumably for litigation purposes. The medical issue being addressed by Grace's comments was whether plaques cause medical problems. Grace's comment stated that "[p]laques have been described in the medical literature as nothing more than 'spots' that can be observed on a lung x-ray, since "by themselves, plaques do not cause loss of function or symptoms." Grace cited several sources, one of which was a quotation from Dr. Murphy who headed the American Thoracic Society Committee that established criteria for diagnosing asbestosis. EPA independently identified the same article, aside from the exhibit by Grace, in its Supplemental Administrative Record No. 2 as 495441. Dr. Murphy, in describing the literature, stated that "[p]hysiologically, hyaline pleural plaques have been referred to as epidemiologic fossils, as beauty spots in the roentgenogram (14), or markers of exposure (the term used by Drs. Franzbalu and Lilis) because, by themselves, plaques do not cause loss of function or symptoms." Dr. Murphy's reference for the "beauty spots" language was Bohlig. For EPA in its June 4, 2002 Document to say that Grace characterizes the observed abnormalities in the screening study as "beauty spots" is a gross mischaracterization of Grace's comments. If EPA is trying to imply that Grace makes light of the findings in the screening study, the implication is incorrect. It is unfortunate that Region 8 tries to divert attention from legitimate scientific and medical issues in this case, including the primary issue whether any exposure pathways existed in 1999 that were creating unacceptable risks.

Indeed, many of EPA's documents in the administrative record support the lack of clinical finding associated with pleural plaques. See, e.g., Jones, Progression of Asbestos Radiographic Abnormalities: Relationships to Estimates of Dust Exposure and Annual Decline in Lung Function (495542) ("Progression of pleural calcification was not associated with significant declines in lung function."); University of Minnesota, Asbestos Health Effects (338062) ("In general, these limited pleural changes and plaques are not associated with clinical and functional abnormalities. They are simply markers of asbestos exposure."); Rey, Environmental Pleural Plaques in an Asbestos Exposed Population in Northeast Corsica (1993) (338255) ("There is no evidence, however, that pleural plaques are precancerous lesions or that they are a risk factor for pleural mesothelioma"); Epler, Asbestos-Related Disease from Household Exposure (1980) (495426) (Plaques can be seen after slight or very brief contact; they can occur from neighborhood exposure in persons living near asbestos mines or factories, in agricultural workers tilling sod containing asbestiform minerals, and in those unknowingly using asbestos containing paints; plaques are not precancerous lesions nor do they cause functional impairment . . ."); deKlerk, Natural History of Pleural Thickening After Exposure to Crocidolite (1989) (459471) (Pleural plaques were not seen to progress beyond their initial thickness or

found that "[o]f those with lung function testing, 2.2% of men and 1.6% of women had restrictive breathing capacity."[44] The low percentages found were associated with occupational exposures, having had chest surgery, being older, having had a high BMI, and being a past or current smoker. Dr. Hughson reports that the screening study does not indicate that pleural abnormalities increased the risk of pulmonary restriction.

Region 8's only support in Libby for progression is a draft unpublished report,[45] entitled Asbestos Related Pleural Disease Due to Tremolite Causes Progressive Loss of Lung Function," by Dr. Alan Whitehouse (495889). According to the draft, 92% of the patients addressed by this draft paper were former employees of Grace or family members of Grace. As set forth in Dr. Hughson's report, Dr. Whitehouse does not provide specific information regarding the 10 patients who did not have the occupational or associated exposures through household contact. Therefore, it is impossible to determine whether the clinical course of the 10 patients differed from those with occupational or associated household exposures. Dr. Whitehouse explicitly states that the exposures experienced by these patients occurred between 1950 to 1975. He further states that "exposures have been ongoing to at least the early 1990s," and notably does not state that exposures were occurring in 1999. We presume that any patients of Dr. Whitehouse who were not exposed occupationally or by associated household exposure have

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extent; evidence indicates that pleural thickening not likely to progress sufficiently to cause impairment of lung function in the absence of parenchymal fibrosis or the occurrence of pleural effusion). See also Harber, Pleural Plaques and Asbestos-Associated Malignancy (1987) ("Any apparent association between plaques and cancer is spurious, being a consequence of their associations with asbestos exposure") (attachment 8).

44 ATSDR, Briefing Materials for the Community (495416).

45 Dr. Hughson's and Dr. Moolgavkar's reports identify several issues and problems with Dr. Whitehouse's report that would be raised in any peer review.

been addressed in the non-occupational case series.[46] As explained above, the case series does not provide evidence that any exposure pathways existed in current day Libby that would result in asbestos-related abnormalities.

Dr. Whitehouse reports that 67 of 123 patients who constituted the study population have no evidence of interstitial disease identified on a chest x-ray or high resolution CT scanning. His draft finds a statistically significant annual loss of lung function, based on the average of the difference between initial and final pulmonary function measurements taken on 123 patients. Dr. Moolgavkar's and Dr. Hughson's reports identify four fundamental problems with Dr. Whitehouse's methodology.

First, the analyses fail to use individual level longitudinal data. A better way to analyze the data would be to consider each response separately and to address the correlations between consecutive readings by using standard statistical techniques. The study could have been set up as a regression problem in which pulmonary capacity is modeled as a function of time depending on a number of covariates, such as age, sex, smoking history, obesity, and most importantly, history of exposure to asbestos. The coefficients of such a model could be estimated using generalized estimating equations (GEE) techniques.

Second, because Dr. Whitehouse uses averages, the decline in pulmonary function which the draft states is statistically significant, could be due to a few outliers in the data, with the majority of individuals showing little or no decline in function except that attributable to aging.

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[46] Of course, Grace cannot confirm this fact because, once again, it has no information about these patients other than what is set out in Dr. Whitehouse's draft and has otherwise been provided with inadequate information to comment effectively on the case series or Dr. Whitehouse's draft.

Third, Dr. Whitehouse does not correlate decline in pulmonary function with asbestos exposure in a quantitative fashion. Therefore, one cannot conclude that the decline is related to exposure, much less to exposure that occurred after the mine closed in 1990.

Fourth, Dr. Whitehouse uses a Sensormedics model 6200 to do the pulmonary function measurements before 1988 and a Medgraphics model 1085 since that time. His draft does not give any indication of the number of patients whose initial and final measurements were made on different machines and whether any attempt was made to calibrate the machines.

Dr. Hughson's report also explains why the findings of Dr. Whitehouse's cannot be applied to the general Libby population. Dr. Whitehouse's draft report describes a group of patients from a non-random sample of people from Libby. As such, they are simply a group of case reports, and cannot be used to infer a poor prognosis for the Libby population.

Aside from the draft Whitehouse Paper, Region 8 points to studies that are far from clear in supporting progression. EPA's citations to Viallat[47] in the May 2, 2002 Amendment do not really support progression. The Supplemental Administrative Record No. 2 contains two documents by Viallat. The first, Environmental Pleural Plaques in an Asbestos Exposed Population in Northeast Corsica (338255), involved a study of residents of an area with naturally occurring surface deposits of chrysotile and tremolite amphiboles. The study, which identified bilateral pleural plaques in 41 percent of the population, stated that "[t]he presence of pleural plaques indicates previous exposure to asbestos only, but not disease." The study also said that "[t]here is no evidence, however, that pleural plaques are precancerous lesions, or that they are a risk factor for pleural mesothelioma." The second study, "Pleural Effects of Environmental

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[47] Grace has already commented on Erlich and Cookson in its December 21, 2001 comments, and cannot find Shephard in the Administrative Record. Grace also cannot find Viallat (1983) in the Administrative Record.

Asbestos Pollution in Corsica" (495546), concluded that the data were insufficient to conclude that either chrysotile or tremolite is responsible for pleural pathology encountered in Corsica. Therefore, neither Viallat study demonstrates, in Region 8's words, "the relationship between the findings of asbestos-related pleural and interstitial abnormalities and serious progression of chronic asbestos-related diseases."

The papers cited by Region 8 in its June 4, 2002 Document do not exhibit anything close to the absolute certainty alleged by Region 8 regarding associations between pleural plaques and functional impairment[48] and are not really "recent" papers. More importantly, they relate to

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[48] Bourbeau, et al., The Relationship between Respiratory Impairment and Asbestos-related Pleural Abnormality in an Active Work Force (1990) (includes citations to studies and an aspect of the study "that supports the clinical opinion that pleural plaques are little more than a sign of asbestos exposure.") (495487).

Hilt, et al, Lung Function and Respiratory Symptoms in Subjects with Asbestos Related Disorders: A Cross-Sectional Study (1986) (495442) (recognizing that "[l]ung function impairment and respiratory symptoms in association with pleural plaques have been a more controversial issue [than diffuse pleural thickening]" and stating that "[a]s the methods applied were crude and the observed differences small, the study does not give conclusive evidence about the prevalence or the degree of lung function disturbances in subjects with different types of asbestos-related disorders.")

Kouris, Effects of Asbestos-Released Pleural Disease on Pulmonary Function (1990) (484312) (stating that "the impact of pleural plaques on pulmonary function has been difficult to evaluate. Two early European studies linked restrictive disease with plaques, but this work was later contradicted by Cotes and, with a much larger study (N=386), by Gaensler, each of whom concluded that plaques had no detrimental effect on lung function.")

Lilis et al, "The Effect of Asbestos-Induced Pleural Fibrosis on Pulmonary Function: Quantitative Evaluation (484266) (stating that "the impact of circumscribed pleural function is still a matter of controversy; differences of opinions among experts are most probably due to the wide possible range of extent and width of circumscribed pleural fibrosis.")

Schwartz, The Clinical Relevance of Asbestos-Induced Pleural Fibrosis (495556) (finding differences were not statistically significant, and that the lung volumes and diffusing capacity were virtually indistinguishable between sheet metal workers with circumscribed plaques and those with normal pleura).Ernest, et al., Pleural Abnormality As A Case of Impairment and Disability (495557) ("[w]hile there is little doubt that bilateral diffuse pleural

occupational cohorts and not to low dose exposures,[49] do not entirely support Region 8's position,[50] and sometimes attribute pulmonary function loss to interstitial fibrosis.[51] Moreover, EPA's June 4, 2002 Document and EPA's administrative record fail to include many of the studies reflecting the prevailing scientific view that asbestosis is necessary to conclude that lung cancer is attributable to asbestos exposure.[52]

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fibrosis may lead to severe impairment, the functional significance of milder degrees of diffuse pleural thickening and especially of pleural plaques remains controversial.")

Hedenstierna, Pleural Plaques and Lung Function in Construction Workers Exposed to Asbestos (1981) (495433) (no significant difference in spirometric test results between smokers with plaques and smokers with neither plaques nor prior asbestos exposure).

Oliver, Asbestos-Related Pleural Plaques and Lung Function (1988) (495454) (pleural plaques in railroad workers associated with a loss of 4.3 percentage point in FVC, but clinical significance of the observed decrement uncertain; also failed to exclude with certainty asbestos induced interstitial fibrosis as a cofounder).

[49] Jarvholm, Pleural Plaques and Respiratory Function (1986) (495440) (showing "slightly impaired lung function" in shipyard workers, but referencing a recent paper showing no difference in respiratory function between persons with and without pleural plaques at an asbestos cement factory, stating that these cement factory workers "probably have much lower exposure to asbestos than shipyard workers," and a "causal or indirect association between pleural plaques and respiratory function is probably strongest in the highly exposed groups").

[50] McGavin, Diffuse Pleural Thickening in Asbestos Workers: Disability and Lung Function Abnormalities (1984) (487091) ("[p]laques have little effect on lung function and seldom cause disability, whereas diffuse pleural fibrosis has been associated with significant impairment of function")

Hillerdal, Asbestos-Related Lesions of the Pleura: Parietal Plaques Compared to Diffuse Thickening Studied with Chest Roentgenography, Computed Tomogrophy, Lung Function, and Gas Exchange (1990) (495489), ("[parietal pleural plaques] [b]y themselves . . . are not considered to affect lung function. Parietal plaques are a frequent finding in an industrialized society.)

[51] Schwartz, Asbestos-Induced Pleural Fibrosis and Impaired Lung Function (1989) (484312) (speculated that subclinical alveolitis or interstitial fibrosis not detected by routine chest radiograms is responsible for the development of restrictive lung function among those with asbestos-induced pleural fibrosis.")

[52] Region 8's administrative record fails to include many of these articles even though they were produced by the United States in the litigation. See Hughes and Weill, Asbestosis is a

Region 8's propensity for attacking every position taken by Grace, no matter what it is, is also evidenced by its critique of Dr. Gaensler, who in fact is an authority on asbestos-related disease, and is cited in many of the scientific articles included in the administrative record.[53] It is odd that Region 8 would criticize Dr. Gaensler's paper submitted by Grace in its comments as being "non-peer reviewed" and "non-published" when EPA relies so heavily on the non-peer reviewed and non-published draft study by Dr. Whitehouse.[54]

Incidence of Mesothelioma in Libby

EPA has included in the Supplemental Administrative Record No. 2 a list of "known mesotheliomas." The death certificates of two of these individuals raise the issue of whether the

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Precursor of Asbestos Related Lung Cancer: Results of a Prospective Mortality Study (1991) ("Because detectable asbestosis is not likely to result from current occupational and general environmental asbestos exposures, the prevention of the effect of exposure on lung fibrosis is likely also to prevent the excess risk of lung cancer. . . . Finally, these data provide further evidence to support the common practice of attributing lung cancer to exposure to asbestos only if asbestosis is present; otherwise, these tumors are, in most cases, due to cigarette smoking") (attachment 9); Jones, Hughes, and Weill, Asbestos Exposure, Asbestosis, and Asbestos-Attributable Lung Cancer (1996) (while the issue of whether asbestosis is a necessary precursor to asbestos-attributable lung cancer cannot at this time be considered settled, the weight of the available evidence strongly supports this proposition) (attachment 10) See also Sluis Cremer, Relation between Asbestosis and Bronchial Cancer in Amphibole Asbestos Miners (1988) (attachment 11); Weiss, Asbestosis: A Marker for the Increased Risk of Lung Cancer Among Workers Exposed to Asbestos (1999) (attachment 12); Weiss, To the Editor (1994) (attachment 13)

[53] EPA June 4, 2002 Document at p. 20; 484267, 484309, 487149, 487155, 495422, 495432, 495462.

[54] Another example of EPA's propensity is Region 8's attacks on Grace's comment that "the ATSDR screening was not diagnostic of asbestos-related disease." The B reader form itself makes this statement and Dr. Lybarger stated that ATSDR was doing follow-up "to review the physician diagnosis for each of the participants in the testing to have an abnormality." (495775) After the 2002 Asbestos Conference in Missoula, Dr. Lybarger is quoted as saying that "the screenings do not confirm asbestos-related diseases." See also St. John's Lutheran Hospital's Press Release, dated April 10, 2000 (484979) (" . . . this phase of the screening will not provide a definitive diagnosis to prove that a person has an asbestos related disease").

cause of death was in fact mesothelioma. In addition, most of the individuals on the list were workers with high occupational exposures, which has no relevance to current day Libby.

The information available to Grace about three of the non-workers cases, Carol Gerard, Darlene Toni Riley, and Reta Orem, involved alleged heavy exposures from pathways which did not exist in current day Libby. Ms. Gerard has alleged exposures by being in contact with mine workers clothes and shoveling truckloads of vermiculite. Ms. Riley alleged exposures from her uncles who worked at the mine, from dusty clothes at her grandmother's house, from playing on the Zonolite property, including on piles of vermiculite wastes, and from popping vermiculite. Mr. Orem's father worked at the mine, but he has also alleged exposure to asbestos from many other companies while working as in insulator in various powerhouses, shipyards, steel mills, refineries, paper mills, chemical plants and/or other facilities. (see attachment 14).

Grace does not have information about Victoria Skidmore and Ford Wilson, even though Grace has requested that information from the United States, and therefore Grace cannot comment on those cases at this time.

EPA points to articles associating pleural plaques with an increased risk of mesotheliomas, but other studies show no association.[55]

Sensitive Population

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[55] See e.g., Pampalon, Environmental Pollution by Asbestos and Public Health in Quebec (495434) ("The present study does not detect any significant difference of mortality between women of asbestos producing cities and women of other cities in Quebec. Accordingly, it does not offer any evidence of an effective contribution by environmental pollution by asbestos to the mortality of the general population."); Harper, Pleural Plaques and Asbestos-Associated Malignancy (1987) ("this study found no association between pleural plaques and asbestos-related malignancies that were independent of other causative factors, such as durations of exposure, age, and cigarette smoking") (attachment 8)

Dr. Anderson's report also addresses EPA's statement in its May 2, 2002 Amendment that EPA must take additional action because "people in Libby have been exposed to amphibole asbestos via multiple pathways, and that cumulative exposures likely contribute to the observed asbestos-related health effects," and "that the continued exposure of this population to any single amphibole asbestos release may further impact their health."[56] Region 8, however, has admitted that it knows "very little about the cumulative non-occupational exposures experienced among those living in Libby."[57]

EPA further has stated that "[a]sbestos exposures that would present acceptable risks to a healthy population may cause an increase in disease in this highly impacted population." Grace agrees with the aspect of this statement that some asbestos exposures can present acceptable risks. Dr. Hughson's report, however, indicates that the medical literature does not support a notion that previous exposure to asbestos "sensitizes" a person or population. He also explains that the linear no-threshold model does not include a concept of "sensitized" people.

EPA Emergency

Grace has, from the beginning of this matter, asserted that EPA's use of emergency and time-critical authority was arbitrary and capricious.[58] Information in the Supplemental Administrative Record No. 2 further supports that EPA had knowledge of Libby conditions prior

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56 EPA May 2, 2002 Amendment at p. 3.

57 June 4, 2002 EPA Document at p. 22.

58 See September 28, 2000 Grace comments. EPA's June 4, 2002 comment that Grace "assiduously avoids challenging the applicability of the 300.415(b) factors" is simply incorrect.

to 1999, and had more than sufficient time to conduct an appropriate investigation and provide an opportunity for comment prior to undertaking its "emergency" measures.[59]

Product

In the May 2, 2002 Amendment, EPA alleges that "Libby vermiculite was given away informally at processing facilities and not inspected, packaged, labeled, warranted, regulated or sold as a commercial product would be" and "under these unregulated circumstances the vermiculite to be removed from residence and businesses does not constitute a product under CERCLA § 104(a)(3)." Grace disagrees with EPA's allegations. Grace will address this legal issue at the appropriate time before the court.

[59] March 31, 2001 report from Nikki L. Tinsley to Christine Todd Whitman, entitled EPA's Actions Concerning Asbestos-Contaminated Vermiculite in Libby, Montana (495722).